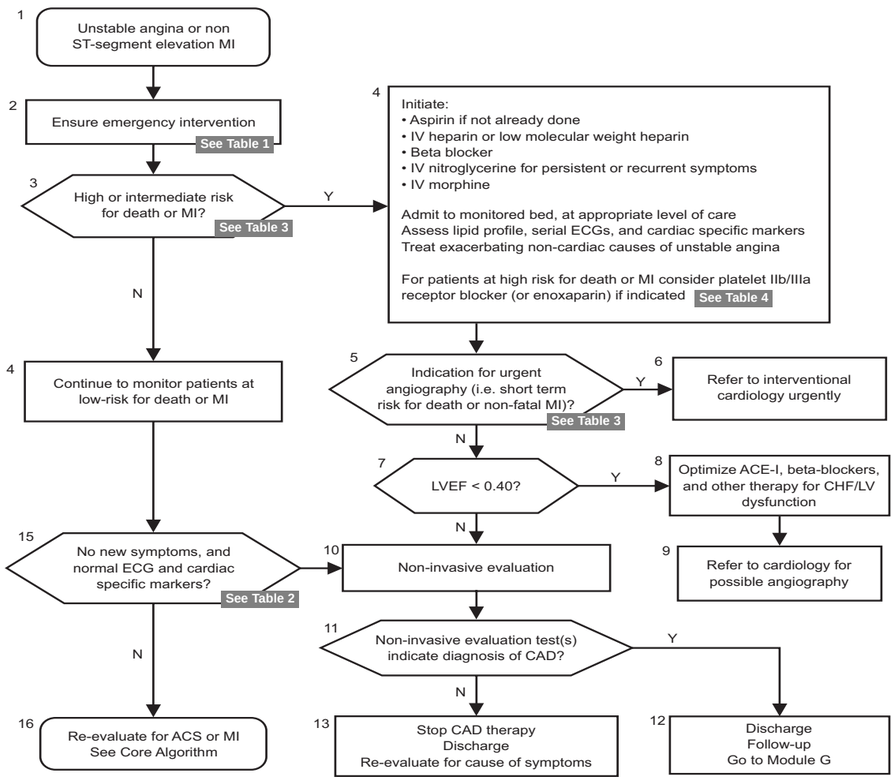
VA/DoD Clinical Practice Guideline Management of Ischemic Heart Disease (IHD) in Primary Care - Module B

Pocket Guide Suspected Acute Coronary Syndorm Unstable Angina/NSTEMI

For initial Evaluation – CORE, Management of AMI, and Follow-Up of Patient with IHD, See Respective Pocket Guides



VA access to full guideline: http://www.oqp.med.va.gov/cpg/cpg.htm
DoD access to full guideline: http://www.cs.amedd.army.mil/Qmo
Sponsored & produced by the VA Employee Education System in cooperation with the Offices of Quality & Performance and Patient Care Services and Department of Defense.

April 2002

MANAGEMENT OF UNSTABLE ANGINA / NSTEMI

1. Ensure emergency interventions for patients who do not meet criteria for emergent reperfusion therapy.
2. Assess short-term risk of death or MI See Table 3

High-Intermediate Risk

Admit to a monitored bed, at appropriate level of care.
Initiate IV heparin or enoxaparin.

High-Risk

Consider GP IIb-IIIa inhibitor therapy.
Refer to urgent angiography, if indicated.

Low-Risk

Monitor cardiac rhythm and serum markers for at least 6 to 8 hours.
Re-evaluate for ACS if change in symptoms, ECG, or serum markers.

3. Perform non-invasive evaluation: (cardiac stress test and LV function) in patients not undergoing angiography.
4. Initiate ACE inhibitor therapy if EF < 0.40.
5. Refer to cardiology, if indicated.
6. Optimize pharmacological therapy for ischemia, angina, and CHF.
7. Discharge patient to home with appropriate follow-up.

Table 1: Emergency Interventions

- Rapidly triage patients with possible acute MI or unstable angina to a high-acuity setting for rapid diagnostic evaluation and treatment
- Obtain 12-lead ECG
- Institute advanced cardiac life support (ACLS) if indicated.
- Obtain serum cardiac markers (troponin or CK-MB)
- Perform expedited and focused history and physical examination to elicit characteristics of MI and contraindications to reperfusion therapy.
- Administer:
  - Non-coated aspirin (160-325 mg)
  - NTG (spray or tablet, followed by IV if symptoms persist)
  - Beta-blockers in the absence of contraindications
- Ensure adequate analgesia (morphine if needed)
- Identify and treat other conditions that may exacerbate symptoms
- Institute continuous ECG monitoring
- Determine whether the patient meets criteria for emergent reperfusion therapy

Increased Risk for Complications or Death Following a MI

- Recurrent angina (spontaneous or inducible)
- Congestive heart failure (CHF)
- Polymorphic ventricular tachycardia, ventricular fibrillation, or sustained monomorphic ventricular tachycardia more than 48 hours from presentation
- Prior MI
- Ejection fraction (EF) <0.40
- Associated severe mitral or aortic valvular disease (e.g., aortic stenosis, aortic regurgitation, or mitral regurgitation)

Table 2: Biochemical Cardiac-Markers for the Evaluation and Management of Patients Suspected of Having an ACS, but Without ST-Segment Elevation of 12 Lead ECG (ACC/AHA UA - NSTEMI, 2000)

Marker	Advantage	Disadvantages	Clinical Recommendations
CK-MB	• Rapid, cost-efficient, accurate assays. • Detection of early reinfarction.	• Loss of specificity in the setting of skeletal muscle disease or injury, including surgery. • Low sensitivity during very early MI (i.e., <6 hours after onset of symptoms) or later after onset of symptoms (i.e., >36 hours) and for minor myocardial damage (detectable by troponins).	• Prior standard and still acceptable diagnostic test in most clinical circumstances. • Familiar to the majority of clinicians.
CK-MB Isoforms	• Early detection of MI.	• Specificity profile is similar to CK-MB. • Current assays require special expertise.	• Useful for extremely early detection of MI (i.e., 3 to 6 hours after onset of symptoms) in centers with demonstrated familiarity with the assay technique. • Experience to date is predominantly in dedicated research centers.
Myoglobin	• High sensitivity. • Early detection of MI. • Detection of reperfusion • Most useful in ruling out MI.	• Very low specificity in the setting of skeletal muscle injury or disease. • Rapid return to normal range limits sensitivity, for later presentations.	• Should not be used as the only diagnostic marker, because of a lack of cardiac specificity. • A more convenient early marker than CK-MB isoforms because of greater availability of assays for myoglobin. Rapid-release kinetics make myoglobin useful for the non-invasive monitoring of reperfusion in patients with established MI.
Cardiac Troponins	• Powerful tool for risk stratification. • Greater sensitivity and specificity than CK-MB. • Detection of recent MI up to 2 weeks after onset. • Useful for the selection of therapy. • Detection of reperfusion.	• Low sensitivity in very early phase of MI (i.e., <6 hours after onset of symptoms) and requires a repeat measurement at 8 to 12 hours, if negative • Limited ability to detect the late minor reinfarction.	• Useful as a single test to efficiently diagnose NSTEMI (including minor myocardial damage), with serial measurements; clinicians should familiarize themselves with diagnostic "cutoffs" used in their local hospital laboratory. • Data on diagnostic performance and potential therapeutic implications are increasingly available from clinical trials.

Table 3: Short-Term Risk of Death or Non-Fatal MI in Patients with UA

	High Risk	Intermediate Risk	Low Risk
Feature	*At least 1 of the following features must be present.*	*No high-risk feature, but one of the following features mustbe present.*	*No high- or intermediate- risk feature, but any of the following*
History	• Accelerating tempo of ischemic symptoms in the preceding 48 hours	• Prior MI, peripheral or cerebrovascular disease, or coronary artery bypass graft (CABG) • Prior aspirin use	
Character of Pain	• Prolonged ongoing rest pain (>20 minutes)	• Prolonged rest angina (>20 minutes), now resolved, with moderate or high likelihood of coronary artery disease (CAD) • Rest angina (<20 minutes or relieved with rest or sublingual NTG)	• New-onset CCS Class III or IV angina in the past 2 weeks without prolonged rest pain (>20 minutes), but with moderate or high likelihood of CAD
Clinical Findings	• Pulmonary edema, most likely related to ischemia • New or worsening mitral regurgitation (MR) murmur • S3 or new/worsening rales • Hypotension, bradycardia, or tachycardia • Age>75 years	• Age >70 years	
ECG Findings	• Angina at rest with transient ST-segment changes >0.05 mV • BBB, new or presumed new • Sustainedventricular tachycardia	• T-wave inversions >0.2 mV • Pathological Q-waves	• Normal or unchanged ECG during an episode of chest discomfort
Cardiac Markers	• Markedly elevated (e.g., TnT or TnI >0.1 μg/mL)	• Slightly elevated (e.g., TnT >0.01, but <0.1 μg/mL)	• Normal

Table 4: FOR SHORT-TERM HIGH RISK PATIENTS
Criteria for Considering Use of Glycoprotein IIb/IIIa Inhibitors

Predictor Variables: Add 1 point to score for every variable (Maximum score = 7)

1	Age >65 years
2	At least 3 risk factors for CAD (smoking; hypertension, hyperlipidemia, diabetes; family history of CAD)
3	Significant CAD (prior coronary stenosis ≥ 50%)
4	ST-deviation (ST depression ≥ .05 mV)
5	Two or more anginal events in the last 24 hours
6	Elevated serum cardiac markers
7	Use of aspirin in the preceding 7 days

Score ≥ 3 Use enoxaparin or glycoprotein IIb/IIIa inhibitor, plus unfractionated heparin
Score < 3 Use enoxaparin or unfractionated heparin